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Key messages

● The Lothian Birth Cohorts of 1921 (N = 550) and 1936 (N = 1091) have cognitive test data from age 11 and old age and are used to examine the contributions to lifetime cognitive change and cognitive change within old age.

● Both cohorts have a range of other ageing-related outcomes—psychosocial and bio-medical—and a wide range of potential predictor variables, including demographic, psychosocial, lifestyle, bio-medical, and genetic.

● Both cohorts have genome-wide SNP testing, and Lothian Birth Cohort 1936 participants have had a detailed structural MRI brain scan especially aimed at examining brain white matter integrity.

Summary

This cohort profile describes the origins, tracing, recruitment, testing, and follow-up of the University of Edinburgh-based Lothian Birth Cohorts of 1921 (LBC1921; N = 550) and 1936 (LBC1936; N = 1091). The participants undertook a general intelligence test at age 11 years and were recruited for these cohorts at mean ages of 79 (LBC1921) and 70 (LBC1936). The LBC1921 have been examined at mean ages of 79, 83, 87, and 90. The LBC1936 have been examined at mean ages of 70 and 73, and are being seen at 76. Both samples have an emphasis on the ageing of cognitive functions as outcomes. Because they have childhood intelligence test scores, the cohorts' data have been used to search for determinants of lifetime cognitive changes, and also cognitive change within old age. The cohorts' outcomes also include a range of physical and psychosocial aspects of wellbeing in old age. Both cohorts have a wide range of variables: genome-wide genotyping, demographics, psychosocial and lifestyle factors, cognitive functions, medical history and examination, and biomarkers (from blood and urine). The LBC1936 participants also have a detailed structural MRI brain scan. A range of scientific findings is described, to illustrate the possible uses of the cohorts.

How did the study come about?

The Lothian Birth Cohorts of 1921 and 1936 are follow-up studies of the Scottish Mental Surveys of 1932 and 1947.[1-5] The surveys had, respectively, tested the intelligence of almost every child born in 1921 or 1936 and attending school in Scotland in the June of those years. Therefore, tracing, recruiting and re-testing people who had taken part in the Surveys offered a rare opportunity to examine the distribution and causes of cognitive ageing across most of the human life course. The

studies described here were initially set up to study determinants of non-pathological cognitive ageing; i.e., the ageing of cognitive functions largely in the normal range, and not principally dementia or other pathological cognitive disorders.

The Lothian Birth Cohort 1921 (LBC1921) was begun in 1999 with the original aim of discovering genetic determinants of cognitive ageing. Funding has been received from the UK's Biotechnology and Biological Sciences Research Council (BBSRC) (wave 1), a Royal Society-Wolfson Research Merit Award to IJD (wave 2), and the Chief Scientist Office (CSO) of the Scottish Government's Health Directorates for wave 3, wave 4 (ongoing throughout 2011), and a questionnaire study between waves 1 and 2.

The Lothian Birth Cohort 1936 (LBC1936) was begun in 2004 with the aim of discovering a wider range of causes of people's differences in cognitive ageing. Funding has been received from Research Into Ageing (wave 1), and Age UK and the UK's Medical Research Council (waves 2 and 3, the latter ongoing throughout 2011 and 2012). BBSRC funded genome-wide genotyping of both cohorts.

What does it cover?

The LBC1921 and LBC1936 studies were set up to discover determinants, beginning with a genetic emphasis, of individual differences in cognitive changes from childhood to old age, and also within old age. However, the independent (predictor, exposure) and the dependent (outcome) variables were always much broader than genetic and cognitive, respectively. Putative determinants in both cohorts include demographic, social, psychological, medical, physiological, biomedical, and genetic

factors. Outcomes of interest in both cohorts include cognitive, other psycho-social, and medical variables. Brain imaging variables—as outcomes, mediators and predictors—are a major focus of the LBC1936.

The original aim of the LBC1921 study was to seek "Molecular genetic influences on cognitive ageing in healthy old people," which was the title of the BBSRC grant that funded the wave 1 data collection. The objectives were: to test the effect of *APOE* e4 status on lifetime cognitive ageing; to collect social, cognitive, and medical data; and to retain the phenotypic information and DNA in order to test other candidate genes. Subsequent, consecutive BBSRC grants, again aimed at explaining variation in lifetime cognitive ageing, studied the effects of single nucleotide polymorphisms (SNPs) from over 100 oxidative-stress-related genes, and conducted a genome-wide association study (GWAS). These latter two awards supported work on both LBC1921 and LBC1936. The principal aims of the CSO-funded waves 3 and 4 of the LBC1921 were to test the common cause hypothesis of cognitive ageing: i.e., they aimed to use the longitudinal data collected within old age to test the idea that age-related physical and cognitive changes proceed in parallel, owing to shared causes.

The original aim of the LBC1936 study was to seek, "Determinants of normal cognitive ageing in survivors of the Scottish Mental Survey 1947," which was the title of the Research into Ageing grant that funded the wave 1 data collection. Those determinants of lifetime cognitive ageing were to include, "genetic, information processing, medical, economic, and psychosocial factors." Wave 2 of LBC1936 was intended, "to identify the mechanisms that cause white matter damage and how this

causes cognitive decline with ageing". New to wave 2 was that the LBC1936 subjects underwent detailed structural magnetic resonance brain imaging (MRI).

From the beginning, the LBC1921 and LBC1936 studies were used to test hypotheses beyond those for which the core grant funding was obtained. Thus, the principal investigators, their research staff and students, and collaborators have used the two cohorts to test a wide range of possible contributors to cognitive changes across the lifespan and within old age, to study ageing and its casual factors more generally, and to address sundry topics for which the cohorts had relevant data.

Who is in the sample?

The Scottish Mental Survey 1932 took place simultaneously across schools in Scotland on June 1st 1932, and tested a total N = 87,498.[3] Only a very few of these children were tested on subsequent days. The test used was a version of the Moray House Test Number 12, a general intelligence test also known as a 'verbal reasoning' test. Almost 70 years later, individuals who had potentially taken part in the survey were identified in Edinburgh and the surrounding area (the Lothians) and invited to participate in the LBC1921. Participants were identified using the Community Health Index (lists of individuals registered with a General Practitioner, which were searched for individuals born in 1921) or through media advertisements. The Community Health Index identified 1120 potential participants. From those invited, 728 responses were received, of which 501 were eligible. The media advertisements generated 423 enquiries, of which 368 were eligible. In total, 550 participants, 234 men and 316 women, joined the LBC1921 and completed wave 1 assessments between 1999 and

2001. They were a mean age of 79 years old. The detailed tracing, recruitment and testing of the LBC1921 are described elsewhere.[1]

The Scottish Mental Survey 1947 used the same intelligence test as the 1932 Survey and took place on June 4th 1947, testing a total N = 70,805.[4] Between 2004 and 2007, individuals from Edinburgh and the Lothians who might have taken part in the 1947 survey were invited to participate in the LBC1936. The Community Health Index was used to identify 1936-born individuals, supplemented by media advertisements. The Community Health Index identified 3810 potential participants, and 3686 were invited. Overall, 2318 responses were received, of which 1226 were interested and eligible (97 of these came from the media advertisements). In total, 1091 participants joined the LBC1936 and were assessed at wave 1 (548 men and 543 women). The detailed tracing, recruitment and testing of the LBC1936 are described elsewhere.[5]

How often have they been followed up, and what is attrition like?

At each wave of testing after the first, only those available participants who had completed all previous waves were invited. This was done because the main reasons for attrition were death, chronic incapacity, and permanent withdrawal. Those with a temporary illness or other reason for unavailability were re-contacted and seen at a later, more appropriate time where possible. Since recruitment and assessment at wave 1 (mean age 79), the LBC1921 have completed 2 further waves of testing, at mean ages 83 and 87. At age 83 (wave 2), 454 participants were invited to participate, and 321 were assessed (145 men and 176 women). Wave 2 ran from 2003 to 2005.[6] For wave 3, running from 2007 to 2008 when the participants were aged 87, 268 participants were invited and 196 attended the research clinic. Those participants

unable to attend the clinic were offered a home visit, and 11 participants were assessed in this way. A further 30 participants were able to supply self-report questionnaire data only. The total sample at wave 3 was 237 (109 men and 128 women).[7] A 4th wave is taking place in 2011, and participators are assessed as near to their 90th birthday as practicable. Some participants have been seen in residential accommodation (waves 3 and 4), and those with possible dementia have been assessed by a geriatric physician at wave 4. The waves of assessment in the LBC1921 are summarised in Table 1, with details of mean ages, numbers and sample composition.

Since recruitment and assessment at wave 1 (mean age 70), the LBC1936 participants have completed a 2nd wave of follow-up at mean age 73. Between 2007 and 2010, 1062 participants were invited and 866 were assessed (448 men and 418 women). The majority of these undertook a detailed structural MRI brain scan at wave 3, and this is being repeated at wave 3. The LBC1936 are attending a 3rd wave of assessment which commenced in 2011. The waves of assessment in the LBC1936 are summarised in Table 1.

With regard to how attrition affects some key variables, the differences between returnees and non-returnees in the LBC1921 and LBC1936 are shown in Table 2. For both LBC samples, ongoing linkages to mortality data were begun at the completion of wave 1. Participants' deaths are routinely flagged and details are retuned to the research team. The LBC1936 are also flagged for morbidity data, using the Scottish Morbidity Records system.

What has been measured?

Cognitive ageing is the central interest in the LBC studies. At all waves, a core cognitive test battery has been completed. LBC1921 and LBC1936 have measures of reasoning, processing speed, executive function, and memory. In addition, detailed medical history and physical information are collected at each wave. The physical data were collected by trained research nurses. Brain imaging data (LBC1936 waves 2 and 3 only) were collected by specialist radiographers. The cognitive and other data were collected by psychology graduates and post-docs. Each wave of assessment involves about a half-day visit to a clinical research facility with one-to-one testing. The LBC1936 brain imaging is performed in a separate clinic visit. Each wave also comprises a substantial set of questionnaires that are completed at home.

In the LBC1921, cognitive ability data were collected at all waves, including repeating the Moray House Test at waves 1 and 3 (previously completed at age 11 years). The cognitive battery consisted of a core set of psychometric tests, supplemented at subsequent waves by tests of processing speed. At each wave, medical history data were collected and updated, and a physical examination was conducted (wave 2 had a more limited physical assessment). At wave 2, LBC1921 participants additionally had retinal photographs taken, and facial photographs for asymmetry measurements. At wave 3, more detailed bodily measures were taken to assess fluctuating asymmetry. Blood samples were taken for DNA extraction and basic biochemistry and haematology (not wave 2). Latterly, biomarkers of inflammation and oxidative stress have been assessed (wave 3 in LBC1921). All cognitive and physical measures are to be repeated at the planned 4th wave of data

collection. Table 1 gives a summary of the LBC1921's assessments across the waves of testing.

The LBC1936 completed a much more detailed battery of psychometric cognitive tests, covering the domains of processing speed, memory, executive function, reasoning, and vocabulary. This was completed at waves 1 and 2. The Moray House test was also included at wave 1. A detailed medical history and physical examination was completed at both waves, and blood samples were taken for DNA extraction, basic biochemistry and haematology and, at wave 2, for assessments of markers of inflammation, oxidative stress, and blood-brain barrier integrity. At wave 2, the LBC1936 had retinal photographs taken, and underwent detailed magnetic resonance brain imaging and Doppler ultrasound scanning of the carotid arteries. All cognitive, physical and brain imaging assessments are being repeated at wave 3. Details of the measures completed at each wave are in Table 1.

Both cohorts have completed self-report questionnaires at each wave. These have provided details of parental background, their children's education and occupation, and their own personality, well-being, social support, and activity participation (the latter 3 have been collected on more than 1 occasion).

What has it found? Key findings and publications

Between them, the LBC1921 and LBC1936 participants' data have appeared in over 100 peer-reviewed publications, so it is not possible to give more than an indicative summary here. Our book—*A Lifetime of Intelligence*—includes a summary and discussion of some of the studies published from the LBC1921 up to the end of 2007.[2]

Given the unusual separation in time between the original intelligence test at age 11 and repeat testing in old age, a central contribution of the LBC studies has been to demonstrate that about half of the variance in cognitive ability is common to the measurements separated by seven decades.[1,7] The studies have examined a wide range of variables to discover the determinants of lifetime cognitive change (the other half of the variation, if we ignore error of measurement for the moment), and the determinants of cognitive change and other aspects of well-being within old age.

An early contribution to cognitive ageing was to show—in the LBC1921—that possession of the e4 allele of the *APOE* gene was associated with intelligence at age 79 but not at age 11.[8] people with the e4 allele decreased in intelligence more than non-carriers across that 68-year period. *APOE* variation continues to influence cognitive ageing across the ninth decade; those individuals with the e4 allele declined more in non-verbal reasoning and verbal declarative memory from age 79 to age 87 than those without the e4 allele.[9] Both LBC studies have contributed to many candidate gene studies of cognitive ability and cognitive ageing.[10-20] Some of these putative associations—typical of candidate gene studies more generally—are failures to replicate. This is why we conducted a study with the LBC1936 sample in which many candidate genes for cognitive ageing were tested together, and few held up, though variants in *COMT*, *KL*, *PRNP*, *PPP1R1B*, *SORL1* and *WRN* merited further study as possible contributors to cognitive ability and change.[21] The cohorts' data have also been used to examine gene systems with respect to cognitive ageing. In one study, over 100 genes associated with oxidative stress and cognition were examined, and an intronic SNP in the *APP* gene was possibly associated with cognitive ageing.[22] Investigation of Alzheimer disease genes, in addition to *APOE*, suggested that a

haplotype from *TRAPPC6A* might be associated with non-verbal reasoning in non-demented older adults.[23] Studies of telomere length in the LBC samples provided limited evidence for association with cognitive or other age-related traits,[24,25] with the possible exception of ischaemic heart disease.[26]

The LBC comprised almost half of the participants used to conduct the first GWAS study of cognitive function in old age.[27] This did not find any SNPs that had genome-wide significant associations with cognitive abilities. However, a gene-based test found that variation in the gene *FNBP1L* was associated significantly—at the genome-wide level—with fluid intelligence in old age. Furthermore, the study used a new method to estimate the 'relatedness' of unrelated subjects based on SNP similarity across 500,000+ SNPs. Using this information, it found that 40% of the variance in crystallised intelligence and 51% of that in fluid intelligence could be accounted for by unknown causal genetic variants in linkage disequilibrium with the tested SNPs. This was the first time that the heritability of cognitive traits had been estimated from DNA testing and not from twin or adoption studies.

Both LBC studies are available to take part in non-cognitive GWAS consortia, and already do so in studies of, for example, personality traits.[28,29] Both LBC studies have also contributed to GWAS discoveries for several biomedical traits, including 68 genetic loci with new gene functions in platelet formation and megakaryopoiesis,[30] 16 new genetic loci influencing lung function,[31] 6 loci affecting mean arterial pressure[32], and a new and strongest genetic influence (*F3*, coagulation factor III) for D-Dimer levels[33]. The LBC studies' data, alone, discovered three single nucleotide polymorphisms that accounted for 18% of the variance in activated partial

thromboplastin time, an important measure of clotting.[34] They are also available to be used as healthy controls for some medical disorders in GWAS studies, and have done so already, e.g. for macular degeneration,[35] severe mental illnesses,[36] cancer[37] and stroke.[38]

Several medical and biomedical factors have been studied with respect to lifetime (childhood to old age) cognitive ageing, including fitness,[39] medications,[40] vitamin B12 and folate,[41] renal function,[42] retinal vasculature network geometry,[43] inflammatory markers,[44] and body and facial symmetry.[45] The case of physical fitness is interesting. In the LBC1921, a fitness variable that was a linear combination of lung function (FEV_1), grip strength, and 6 metre walk time, accounted for over 3% of the variance in cognitive change between age 11 and age 79.[37] Also in the LBC1921, grip strength and non-verbal reasoning had cross-sectional associations at ages 79, 83, and 87; however, their age-related slopes were not correlated, and there was no evidence for any reciprocal dynamic influence over time.[46]

Brain imaging-derived variables that have been studied and found to be associated with cognitive function in old age include white matter lesions, white matter integrity as assessed using diffusion tensor- and magnetisation transfer-derived variables, tractography-based white matter integrity, and brain iron deposition.[47-51] A detailed protocol paper is available that addresses the rationale and methods used in the extensive structural magnetic resonance imaging scan that is undertaken by members of the LBC1936.[52]

Psycho-social variables that have been studied as determinants of lifetime cognitive change include dietary and lifestyle factors such as caffeine,[53] B vitamins, antioxidant and flavonoid intake,[54,55] smoking,[56] alcohol,[57] body mass index,[58] social and intellectual engagement,[59] and childhood intelligence level.[60] The discovery of birth and placental weights and other birth data in some of the LBC1921 has produced some evidence of lifecourse continuity between these and brain white matter integrity in old age.[61]

Two summary points may be made. Most putative predictors make small contributions to the variation in lifetime cognitive change: around 1 to 2% is typical. Second, there is often reverse causation (or confounding), because we have found that associations between contemporaneously-assessed cognitive functions and putative determinants in old age are markedly attenuated—sometimes to below conventional statistical significance—after adjusting for childhood intelligence. To offer some different examples for the two cohorts, this occurred with apparent contributions from intellectual engagement,[59] C-reactive protein,[44] and brain iron deposits[51]: after adjustment for childhood intelligence, these contemporaneous associations with cognitive ability in old age were markedly attenuated or disappeared entirely.

Because there is such a wide range of phenotypic assessment in the LBC studies, they were always intended to be used to examine broader aspects of ageing than just the cognitive. Thus, they have been used to study outcomes such as personality and mood symptoms,[62,63] processing speed as ageing biomarkers,[64] testing the hierarchical psychometric structure of cognitive abilities,[65] blood pressure,[66] renal function,[67] height and its association with cognitive ageing,[68] predictors of tooth loss in old age,[69]

how haematological profiles differ between men and women in old age,[70] lifetime and cross-generational social mobility,[71,72] activities of daily living,[73] determinants of life satisfaction in old age,[74] and associations with other aspects of health and well-being in older people.[75] They have made contributions to validating cognitive instruments to estimate prior cognitive ability in dementia.[76]

What are the main strengths and weaknesses?

The main, unusual strength of both cohorts is the possession of a valid intelligence test score from age 11 years in people who are in old age, and who then took the same intelligence test as well as a host of other cognitive and other tests. This, with the detailed follow-up cognitive testing, means that almost life-long cognitive changes can be described and explained. A strength of the cohorts lies in being able to test for possible reverse causation between intelligence and putative determinants of cognitive ageing. It is also a strength that both LBC studies are narrow age cohorts, which means that the variables measured reflect differential ageing and not chronological age, which can otherwise swamp the variance in assessments and inflate effect sizes. Both samples have genome-wide testing on over half a million SNPs, and the LBC1936 has a detailed structural brain imaging protocol, including diffusion tensor and magnetisation transfer imaging. Additional strengths are the wide variety of medical and psycho-social variables that are collected.

Weaknesses include the limited sample sizes of the two cohorts, and their being somewhat restricted in range with respect to childhood intelligence and childhood and adult socio-economic status. The mean (SD) Moray House Test score at age 11 was: 46.4 (12.1) for the LBC1921 compared with 34.5 (15.5) for the whole of Scotland;

and 49.0 (11.8) for the LBC1936 compared with 36.7 (16.1) for the whole of Scotland, and a mean of 40.3 for the Edinburgh region. However, the reports on the samples are able—because the whole populations were tested at age 11—to estimate accurately by how much these factors might attenuate effect sizes. Both are entirely composed of Caucasian participants. Neither sample has, as yet, been fully ascertained for dementia.

Can I get hold of the data? Where can I find out more?

The LBC study data have been the subject of many internal (within the University of Edinburgh) and external collaborations, which are encouraged. The LBC1921 study data have been made available to, and used by, the HALCyon collaboration.[77,78] Those who have interests in outcomes other than cognitive domains are particularly encouraged to collaborate. The current procedure for those who wish to work with the data is initially to email Ian Deary to ask for a 'LBC Data Request Form'. Both LBC studies have clear data dictionaries which help researchers to discern whether the variables they wish to use are present; these provide a simple short title for each variable, alongside a long, common-sense description of each variable. The process is facilitated by a full-time LBC database manager. Such proposals, when approved, are conducted in collaboration with appropriate members of the LBC study team.

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Table 1

The Lothian Birth Cohorts of 1921 and 1936

Cohort	Wave	Mean age (SD)	Sample (men/women)	Measurements
LBC1921	Scottish Mental Survey 1932	10.9 (0.3)	87,498 (44,210/43,288)	Cognitive ability: Moray House Test
	1 (1999-2001)	79.1 (0.6)	550 (234/316)	Cognitive ability: Moray House Test and cognitive test battery consisting of executive function, reasoning and memory. Sociodemographic information: education, occupation, marital status/living arrangement. History of disease and medication use, anxiety and depression, smoking and alcohol status. Physical and fitness measures: disability scale, ECG, height, weight, blood pressure, lung function, grip strength, 6m walk time, visual acuity, dentition. Blood samples taken for DNA extraction, biochemistry and haematology. Self-reported parental background, education and occupation, well-being, social networks and support, activities, and personality.
	2 (2003-05)	83.4 (0.5)	321 (145/176)	Cognitive ability: Wave 1 cognitive test battery plus processing speed. History of disease and medication use. Physical and fitness measures: blood pressure, grip strength, visual acuity. Facial asymmetry photographs, retinal photography. Children's education and occupation. Self-reported retrospective occupational characteristics, social networks and support and activities. Also religious involvement and spirituality.
	3 (2007-08)	86.6 (0.4)	237 (109/128)	Cognitive ability: Moray House Test and Wave 2 cognitive test battery. Sociodemographic information: marital status/living arrangement. History of disease and medication use, anxiety and depression, smoking and alcohol status. Physical and fitness measures: as Wave 1 plus ABPI, but not ECG or dentition. Blood samples as Wave 1 plus markers of oxidative stress and inflammation (plasma and

				serum stored at -80^{o}C). Fluctuating asymmetry measurements. Self-reported well-being and personality.
	4	~90	Commenced January 2011	Core cognitive and physical battery.
LBC1936	Scottish Mental Survey 1947	10.9 (0.3)	70,805 (35,809/34,996)	Cognitive ability: Moray House Test.
	1 (2004-07)	69.5 (0.8)	1091 (548/543)	Cognitive ability: Moray House Test and psychometric battery consisting of executive function, reasoning, memory, and processing speed. Sociodemographic information: education, occupation, marital status/living arrangement. History of disease and medication use, anxiety and depression, smoking and alcohol status. Physical and fitness measures: disability scale, height, weight, blood pressure, lung function, grip strength, 6m walk time, visual acuity. Blood samples taken for DNA extraction, biochemistry and haematology. Self-reported parental background, education and occupation, childrens' education and occupation, well-being, social networks and support, activities, personality and diet.
	2 (2007-2010)	72.5 (0.7)	866 (448/418)	As Wave 1 plus ABPI, balance, dentition, markers of oxidative stress, inflammation, and blood-brain barrier integrity (plasma and serum stored at -80^{o}C), retinal photography, magnetic resonance brain imaging and carotid Doppler ultrasound. No Moray House Test, education or occupation. Self-reported well-being and personality.
	3	~76	Commenced July 2011	Core cognitive and physical battery, repeat MR brain imaging.
Both	Ongoing			All participants are flagged with routine data sources providing deaths since wave 1, and, for LBC1936, morbidity data since wave 2.

Note. For simplicity, the ages at waves 1, 2 and 3 in the LBC1921 are referred to as 79, 83 and 87 respectively. For the LBC1936, waves 1 and 2 are referred to as ages 70 and 73 respectively.

Table 2

Baseline sample characteristics by returning status in the Lothian Birth Cohorts

Sample	Variable	Returnees	Non-returnees
LBC1921	Sex (male)	46.0%	38.9%
	Age-11 IQ	101.6 (14.2)	98.8 (15.5)
	Age-79 IQ	103.6 (12.9	97.3 (15.9)
	MMSE	28.4 (1.5)	28.0 (1.8)
	Father's SES	2.7 (1.0)	2.8 (0.9)
	Participant SES	2.1 (0.8)	2.3 (0.9)
	CVD (yes and unsure/angina only)	29.4%	30.0%
	Grip strength	28.0 (9.6)	25.4 (8.6)
	FEV_1	2.0 (0.6)	1.8 (0.6)
LBC1936	Sex (male)	51.7%	44.4%
	Age-11 IQ	100.7 (15.3)	97.4 (13.4)
	Age-79 IQ	101.0 (14.1)	96.2 (16.5)
	MMSE	28.9 (1.4)	28.6 (1.6)
	Father's SES	2.9 (0.9)	2.9 (0.9)
	Participant SES	2.4 (0.9)	2.6 (0.9)
	CVD (yes)	23.8%	27.6%
	Grip strength[a]	29.4 (10.0)	27.2 (10.6)
	FEV_1	2.4 (0.7)	2.1 (0.7)

Note. Values given are mean and standard deviation, except for sex and CVD, for the wave 1 sample (age 79 in LBC1921 and age 70 in LBC1936) split by returning status. For LBC1921, returnees refers to those participants who attended all 3 waves of assessment (at wave 3, some participants completed self-report questionnaires only). For LBC1936, returnees refers to the participants who attended waves 1 and 2. IQ calculated from Moray House Test score corrected for age in days at time of testing and converted to IQ scale. MMSE = Mini-Mental State

Examination. Father's SES (= socio-economic status) is participant's father's social class when the participant was aged 11. CVD = cardiovascular disease. FEV_1 = forced expiratory volume from the lungs in one second.
[a]Grip strength reported as best of three from the right hand.